

**Plasma N-Terminal Pro-B-Type Natriuretic Peptide as a Long-Term Prognostic Marker After Major Vascular Surgery**

Feringa HH, Schouten O, Dunkelgrun M, et al. Heart 2007;93:226-31.

**Conclusion:** Levels of N-terminal pro-B-type natriuretic peptide (NT-proBNP) predict major adverse cardiac events and long-term mortality after major peripheral vascular surgery.

**Summary:** Risk stratification is widely practiced in vascular surgical patients. Previously identified markers of cardiovascular disease have a mixed track record in predicting major cardiovascular events after vascular surgery. NT-proBNP has recently been shown to have prognostic value in patients with acute coronary syndromes and in those with stable coronary artery disease (Eur Heart J 2005;26:241-9; Br J Surg 2005;92:1041-5). In the current study, the authors sought to determine whether preoperative plasma NT-proBNP levels, when evaluated with conventional cardiac risk factors and dobutamine stress echocardiography (DSE), have long-term prognostic value in patients undergoing major peripheral vascular surgery.

This was a single-center prospective cohort study that involved 335 patients undergoing lower extremity bypass surgery or abdominal aortic aneurysm repair. Baseline NT-proBNP was measured before surgery, and patients were also evaluated using the revised cardiac risk index. DSE was performed routinely. End points included all-cause mortality and major adverse cardiac events (MACE) during follow-up.

Median age of the patients was 62 years, and 76% were male. The median NT-proBNP level was 186 ng/L (interquartile range, 65 to 444 ng/L). Median follow-up was  $14 \pm 6$  months, during which 49 patients (15%) died and 50 (15%) experienced a MACE. Using receiver operator characteristic curves, NT-pro-BNP had greater predictive value for mortality and MACE compared with that of the cardiac risk score and DSE. A NT-proBNP level of 319 ng/L was the optimal cutoff value to predict 6-month mortality and MACE. Adjusting for cardiac risk score, DSE, age, and cardioprotective medications, NT-proBNP  $\geq 319$  ng/L had a hazard ratio of 4.0 for all cause mortality (95% confidence interval [CI], 1.8 to 8.9) and a hazard ratio of 10.9 for MACE (95% CI, 4.1 to 27.9).

**Comment:** Natriuretic peptides are indigenous cardiac hormones. NT-proBNP is synthesized in ventricular muscular and released in response to wall stress (Circulation 1993;87:464-9). Given the relatively poor track record of other markers of cardiac risk in vascular surgical patients, this cardiac hormone deserves additional study as a predictor of perioperative risk and long-term mortality in vascular surgical patients.

**Smoking, Alcohol Consumption, and Raynaud's Phenomenon in Middle-Age**

Suter LG, Murabito JM, Felson DT. Am J Med 2007;120:264-71.

**Conclusion:** Middle-aged men and women have different physiologic mechanisms underlying Raynaud's syndrome. Sex-specific therapeutic approaches may be appropriate for patients with Raynaud's syndrome in middle-age.

**Summary:** Studies examining relationships between smoking, alcohol consumption, and Raynaud's syndrome have produced conflicting results. The authors sought to determine whether alcohol and smoking were independently associated with Raynaud's syndrome in a large community-based cohort. Patients were derived from the Framingham Heart Study offspring cohort. A validated survey to classify Raynaud's syndrome was used. Sex-specific analysis of Raynaud's status was performed according to alcohol consumption and smoking status in 1840 women and 1602 men. The relationship of Raynaud's syndrome to smoking and alcohol consumption was then determined with multivariable logistic regression analysis.

Raynaud's syndrome was not associated with smoking in women but was associated with increased risk in men (adjusted odd ratio [OR], 2.59; 95% confidence interval [CI], 1.11 to 6.04). In women, heavy alcohol consumption, defined as  $>3.5$  ounces of alcohol per week, was associated with an increased risk of Raynaud's syndrome (adjusted OR, 1.69, 95% CI, 1.02 to 2.82). In men, moderate alcohol consumption, defined as  $>1$  but  $<7$  ounces of alcohol per week, was associated with a reduced risk of Raynaud's syndrome (adjusted OR, 0.51; 95% CI, 0.29 to 0.89). In both men and women, red wine consumption was associated with a decreased risk of Raynaud's syndrome (adjusted OR, 0.59; 95% CI, 0.36 to 0.96 in women; adjusted OR, 0.30; 95% CI, 0.15 to 0.62 in men).

**Comment:** The conclusions of this study are probably valid. The authors used a validated survey to classify Raynaud's syndrome, had adequate sample size, and adjusted for confounders in their multivariable analysis. These are probably the best data available regarding the effects of smoking and alcohol consumption with respect to gender in patients with Raynaud's syndrome.

**The Fate of the Distal Aorta After Repair of Acute Type A Aortic Dissection**

Holstead JD, Meier N, Etz C, et al. J Thorac Cardiovasc Surg 2007;133:127-35.

**Conclusions:** Expansion of the distal aorta after repair for acute type A aortic dissection is linear and slow, with a low risk of distal reoperation. The risk of death is twice that of a healthy population.

**Summary:** The natural history of the distal aorta in patients with acute type A aortic dissection is not fully understood. The authors analyzed factors influencing long-term survival, operation of the distal remaining aorta, and segmental growth rates of the distal aorta after successful repair of acute type A aortic dissection. Between 1986 and 2003, 176 consecutive patients (70% male) with a mean age of 60 years underwent repair of acute type A aortic dissection using an open distal anastomotic technique with resection of the intimal flap. Patients had follow-up computed tomography scans with digitalization of distal aortic segments and calculation of segment-specific growth rates of the distal aorta. Factors influencing faster growth were analyzed, and patient survival and the incidence of distal reoperation were also determined.

A total of 89 patients (57%) had sufficient imaging data for calculations of serial growth rates. After repair of the acute type A aortic dissection, the median diameters of the aorta were aortic arch, 3.6 cm; descending aorta, 3.7 cm; and abdominal aorta, 3.2 cm. Respective growth rates were 0.8, 1.0, and 0.8 mm/y. Greater growth rate in the descending aorta was predicted by an initial diameter of  $>4$  cm ( $P = .005$ ) and by diameters of  $<4$  cm with a patent false lumen ( $P = .004$ ). Male sex significantly affected adversely growth rate in the abdominal aorta ( $P = .05$ ).

Sixteen patients had a distal aortic reoperation ( $n = 25$ ). The risk of reoperation was 16% at 10 years. Survival after repair of acute type A aortic dissection was influenced by age ( $P < .0001$ ), neurologic deficit at time of presentation ( $P = .04$ ), lack of preoperative thrombus in the false lumen of the ascending aorta ( $P = .03$ ), and a patent distal false lumen postoperatively ( $P = .06$ ). The site of the operation did not influence long-term survival.

**Comment:** The data regarding expansion rates of the residual aorta after acute type A dissection repair should be viewed with some caution. Only 57% of survivors had sufficient imaging data for calculation of late growth rates. There may have been patients with accelerated growth rates or aortic complications secondary to an accelerated growth rate who were not included in this study. It is probably best to regard the growth rates presented here as "ballpark" figures.

**The Patterns of Communication Breakdowns Resulting in Injury to Surgical Patients**

Greenberg CC, Regenbogen SE, Studdert DN, et al. J Am Coll Surg 2007;204:533-40.

**Conclusion:** Verbal communications between caregivers and surgical attending staff, especially those involving ambiguity about responsibilities, are sources of communication breakdown in the care of the surgical patient.

**Summary:** It is assumed communication breakdowns threaten the safety of surgical patients. There are, however, little data available regarding methods of improving communication in the care of surgical patients. In this study, 444 surgical malpractice claims were reviewed from four liability insurers. From this pool, 60 cases involving communication breakdowns resulting in harm to patients were identified. Cases were analyzed by two surgical reviewers to identify common characteristics. Interventions to prevent communication breakdown were developed, and their potential impact was assessed by the patterns of communication breakdown that were identified.

Eighty-one communication breakdowns were identified in the 60 cases. Communication breakdowns occurred in the preoperative (38%), intraoperative (30%), and postoperative periods (32%). Most communication breakdowns were verbal (92%). Attending surgeons were the most common member of the surgical team involved with communication breakdowns. Factors associated with communication breakdown were ambiguity about responsibility (73%) and status asymmetry (74%). The most common communication breakdown involved residents failing to notify attending surgeons of critical events and failure of attending surgeons to communicate with other attending surgeons during hand-offs. Communication breakdowns occurred during hand-offs in 43% of cases, and 39% of communication breakdowns were associated with transfers in the patient's location within the hospital.

**Comment:** This type of information, on the surface, seems somewhat boring. It is, however, absolutely critical to identify system problems that may adversely affect patient safety. Because the database for this study was malpractice claims, this study obviously reflects only a small number of the communication breakdowns that actually occur. We need mandatory interventions to prevent communication breakdowns involving our surgical patients. Such communications may include mandatory communication with attending surgeons, transfer protocols, structured protocols for hand-offs, and standard use of read-back techniques.

**Non-Viral Gene Transfer of Hepatocyte Growth Factor Attenuates Neurologic Injury After Spinal Cord Ischemia in Rabbits**

Shi E, Jiang X, Kazui T, et al. J Thorac Cardiovasc Surg 2006;132:941-7.

**Conclusion:** Administration of hepatocyte growth factor (HGF) by gene transfer attenuates neurologic injury in an animal model of spinal cord ischemia.

**Summary:** It is known that HGF can function as an angiogenic agent (Gene Ther 2000;7:417-27). It also appears to be a potent neurotropic factor (Eur J Neurosci 2000;12:3453-61). The neuron-protective effects of HGF have led to the thought that it may be beneficial for preventing neurologic defects secondary to ischemic injury of the spinal cord.

The authors used HGF plasmid combined with a hemagglutinating virus of the Japan (HVJ) envelope vector in a rabbit model. The HVJ envelope vector was injected intrathecally. A control vector, or saline, was administered to control animals. Five days after injection, spinal cord ischemia was induced by means of occluding the infrarenal aorta for 30 minutes. Recovery of hind limb motor function was then assessed during a 14-day period using Tarlov criteria. Tarlov criteria quantify hind limb motor function on a scale of 0 to 5, with 0 being no movement and 5 being a normal hop in the rabbit.

HGF was detected in cerebral spinal fluid 3 days after gene transfer, with a peak at 5 days. HGF gene transfer in the experimental animals increased capillary density and gray matter and decreased spinal cord edema compared with controls. At 14 days, all rabbits pretreated with saline or control vector had a Tarlov score of 0. Transfection of HGF before inducing spinal cord ischemia resulted in a Tarlov score of 5 in eight of nine rabbits after 14 days. After euthanizing the rabbits, histologic examination indicated preservation of motor neurons to a greater extent in the HGF gene-transferred rabbits than those administered control vector or saline.

**Comment:** The data indicate gene transfer into the spinal cord can be successful with intrathecal administration of HVJ envelope vector containing an HGF expression plasmid and that administration of HGF before inducing spinal cord ischemia reduces neurologic deficits associated with spinal cord ischemia. The idea of prophylactic administration of HGF gene into the human intrathecal space before thoracoabdominal aneurysm repair to protect against spinal cord ischemia is intriguing.